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## **Effect of maternal infection on progeny growth and resistance mediated by maternal genotype and nutrient availability**

Höckerstedt, Layla ; Susi, Hanna ; Laine, Anna-Liisa

**Abstract:** Maternal effects of pathogen infection on progeny development and disease resistance may be adaptive and have important consequences for population dynamics. However, these effects are often context-dependent and examples of adaptive transgenerational responses from perennials are scarce, although they may be a particularly important mechanism generating variation in the offspring of long-lived species. Here, we studied the effect of maternal infection of *Plantago lanceolata* by *Podosphaera plantaginis*, a fungal parasite, on the growth, flower production and resistance of the progeny of six maternal genotypes in nutrient-rich and nutrient-poor environments. For this purpose, we combined a common garden study with automated phenotyping measurements of early life stages, and an inoculation experiment. Our results show that the effects of infection on the mother plants transcend to impact their progeny. Although maternal infection decreased total leaf and flower production of the progeny by the end of the growing season, it accelerated early growth and enhanced resistance to the pathogen *P. plantaginis*. We also discovered that the effects of maternal infection affected progeny development and resistance through a three way-interaction between maternal genotype, maternal infection status and nutrient availability. **Synthesis.** Our results emphasize the importance of maternal effects mediated through genotypic and environmental factors in long-living perennials and suggest that maternal infection can create a layer of phenotypic diversity in resistance. These results may have important implications for both epidemiological and evolutionary dynamics of host–parasite interactions in the wild.

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## RESEARCH ARTICLE

Journal of Ecology



# Effect of maternal infection on progeny growth and resistance mediated by maternal genotype and nutrient availability

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## Abstract

1. Maternal effects of pathogen infection on progeny development and disease resistance may be adaptive and have important consequences for population dynamics. However, these effects are often context-dependent and examples of adaptive transgenerational responses from perennials are scarce, although they may be a particularly important mechanism generating variation in the offspring of long-lived species.
2. Here, we studied the effect of maternal infection of *Plantago lanceolata* by *Podosphaera plantaginis*, a fungal parasite, on the growth, flower production and resistance of the progeny of six maternal genotypes in nutrient-rich and nutrient-poor environments. For this purpose, we combined a common garden study with automated phenotyping measurements of early life stages, and an inoculation experiment.
3. Our results show that the effects of infection on the mother plants transcend to impact their progeny. Although maternal infection decreased total leaf and flower production of the progeny by the end of the growing season, it accelerated early growth and enhanced resistance to the pathogen *P. plantaginis*.
4. We also discovered that the effects of maternal infection affected progeny development and resistance through a three way-interaction between maternal genotype, maternal infection status and nutrient availability.
5. *Synthesis*. Our results emphasize the importance of maternal effects mediated through genotypic and environmental factors in long-living perennials and suggest that maternal infection can create a layer of phenotypic diversity in resistance. These results may have important implications for both epidemiological and evolutionary dynamics of host–parasite interactions in the wild.

## KEYWORDS

automated phenotyping, host–parasite interactions, maternal effects, perennial plant, *Plantago lanceolata*, transgenerational defence priming

## 1 | INTRODUCTION

Plant pathogens are ubiquitous in both natural and managed ecosystems, and they have complex and diverse effects on their host's survival and reproduction. These effects include host mortality, reduced growth and lower seed production, which is caused

either directly by the pathogen or indirectly due to hampered plant growth (Agrios, 2005; Alexander, 2010; Gilbert, 2002; Jarosz & Davelos, 1995). Natural plant populations support extensive phenotypic variation in disease resistance (Antonovics et al., 2011; Laine et al., 2011; Lebeda et al., 2014). Traditionally, phenotypic variation in resistance is assumed to have a genetic basis (Flor, 1956), where

variation is maintained through a co-evolutionary arms race with pathogens (Bergelson, Dwyer, et al., 2001; Bergelson, Kreitman, et al., 2001). However, we are becoming increasingly aware that the local context—consisting of both abiotic and biotic variables—may alter the resistance phenotype directly and through genotype-by-environment interactions (Blanford et al., 2003; Raghavendra & Newcombe, 2013). To date, remarkably little is known about how the conditions experienced by the parental generation contribute to phenotypic resistance diversity through non-genetic inheritance (Bonduriansky & Day, 2009). In particular, transgenerational priming (Bruce et al., 2007; Luna et al., 2012; Martinez-Medina et al., 2016; Mauch-Mani et al., 2017)—whereby maternal infection renders the offspring more resistant against pathogen attack (Holeski et al., 2012)—could create a layer of phenotypic diversity in resistance to provide further protection from infection.

It is well established that pathogen infection can prime defence plasticity in plant hosts, resulting in enhanced activation of induced defence mechanisms (Bruce et al., 2007; Mauch-Mani et al., 2017). Although often the primed response may be limited to a short time window (Frost et al., 2008; Luna et al., 2012), there is also evidence of a response that is maintained throughout the plant's life cycle (Martinez-Medina et al., 2016; Mauch-Mani et al., 2017). The effects of priming can be passed on to subsequent generations through the embryo via non-genetic mechanisms including DNA methylation, chromatin remodelling and small RNA signalling (Mauch-Mani et al., 2017; Singh & Roberts, 2015), and therefore transgenerational priming represents a type of plant immunological memory (Martinez-Medina et al., 2016). Increased resistance against pathogens in the progeny of infected mothers was first shown in tobacco plants inoculated with tobacco mosaic virus (Roberts, 1983), and later studies mostly performed with short-living annuals and model species have described similar responses (reviewed by Holeski et al., 2012). Results from these studies have confirmed that transgenerational priming may enhance resistance in the progeny of plants exposed to the same or different pathogen species (Kathiria et al., 2010; Slaughter et al., 2012) and hence, maternal effects are often viewed as a form of adaptive, transgenerational plasticity (Agrawal et al., 1999; Frost et al., 2008; Marshall & Uller, 2007; Mauch-Mani et al., 2017; Uller et al., 2013; Yin et al., 2019).

Under pathogen attack, both host growth and reproduction are typically reduced, host resources are allocated to defence and depleted by the parasite, and in some cases hosts are exposed to pathogen-produced toxins (Tsuje et al., 2013; Van Kal, 2006; Yoder, 1980). Hence, transgenerational effects of maternal infection—or other stressors—are not always expected to be adaptive (Burton & Metcalfe, 2014; Marshall & Uller, 2007). In agricultural crops, it is well established that maternal infection may reduce the quantity of the produced seeds (Agrios, 2005; Argyris et al., 2003; Strange & Scott, 2005). In addition to producing fewer seeds, infected plants have been reported to produce smaller seeds, and seeds that have reduced protein content when compared to seeds produced by healthy plants (Edwards et al., 2001; Murray et al., 1995). Parental pathogen infection may also limit the growth in asexually spreading

clonal species (Piqueras, 2001). While such reductions in seed quality due to parental infection are expected to affect seed viability, seedling emergence, growth and reproduction, to date this has not been verified, particularly in natural populations (Burdon & Laine, 2019).

How consistently maternal infection generates variation in offspring fitness and resistance depends on how the effects vary across genotypes and environments. Experiments that focused on maternal effects induced by insect herbivores have shown that maternal effects can be highly genotype specific (Agrawal, 2002; Colicchio, 2017; Holeski et al., 2013), and may vary across genotypes in different abiotic conditions (Münzbergová & Hadincová, 2017; Rendina González et al., 2018; Vivas et al., 2013; Vu et al., 2015). Hence, how strong and durable the pathogen-induced priming effects are, may depend on the genotype-specific response to infection, and the quality and predictability of the environment that both mothers and their offspring encounter (Agrawal et al., 1999; Herman & Sultan, 2011; Kuijper & Hoyle, 2015; Luna et al., 2012; Mauch-Mani et al., 2017).

In this study, we were particularly interested in whether the effects of maternal infection depend on the level of nutrients available to the offspring. Nutrient availability is considered a key determinant of plant growth but may also affect plant disease tolerance and resistance (Agrios, 2005; Amtmann et al., 2008; Dordas, 2008; Mittler & Blumwald, 2010; Suzuki et al., 2014). Generally, nutrient stress is shown to weaken plant defences (Amtmann et al., 2008; Dordas, 2008; Suzuki et al., 2014). However, there is also evidence that increasing nutrients can increase the severity of infection, especially by obligate pathogens (Dordas, 2008), and that the effects of nutrients on disease resistance vary among plant species (Veresoglou et al., 2013) and genotypes (Laine, 2007). Agriculture has replaced natural habitats across the world, and those remaining are increasingly occurring adjacent to agricultural practices (Foley et al., 2005). With both nutrient spillover from agricultural habitats (Matson et al., 1997), and nitrogen deposition Hyvönen et al., 2007), natural populations are increasingly exposed to new levels of nutrients, and there is a pressing need to quantify the consequences of this.

Long-lived perennial plants may face short periods of stress with respect to their life span, and thus transgenerationally induced phenotypic plasticity may be a particularly advantageous strategy to cope with changing conditions (Auge et al., 2017; Donelson et al., 2018). Here, we investigate whether maternal infection by powdery mildew *Podosphaera plantaginis* affects progeny growth, flower production and resistance phenotype in the perennial ribwort plantain *Plantago lanceolata* in a series of experiments described in Figure S1. We also tested whether the effect varies depending on the maternal genotype, and between two contrasting nutrient environments of the offspring. In our study area in the Åland archipelago *P. lanceolata* grows on open dry meadows that are characterized by low in nutrient concentrations. However, soils in populations adjacent to agricultural fields have significantly higher nutrient levels (H. Susi & A.-L. Laine, pers. comm.), with potentially far reaching implications for the

ecology and evolution of these populations. We used seeds from six maternal *P. lanceolata* genotypes that had been clonally propagated into multiple individuals and grown in common garden conditions for 3 years with and without infection. First, we monitored early-life development of the offspring, in two contrasting nutrient environments in the greenhouse conditions using automated phenotyping technology. Subsequent life-history traits were then monitored under the common garden settings. Finally, the progeny resistance against powdery mildew strains was assessed in a laboratory inoculation trial. The epidemic of *P. plantaginis* peaks late in the growing season, and hence we measured the resistance of mature progeny.

## 2 | MATERIALS AND METHODS

### 2.1 | *Plantago lanceolata* and *Podosphaera plantaginis* interaction

*Plantago lanceolata* L., the ribwort plantain, is a perennial monoecious herb (Sagar & Harper, 1964). It is an obligate outcrosser that can reproduce sexually via wind pollination, or asexually by producing vegetative side rosettes (Laine, 2004). Seeds drop close to the mother plant forming a long-term seed bank (Bos, 1992). Obligate biotroph *P. plantaginis* (Castagne; U. Braun and S. Takamatsu), belonging to the order *Erysiphales* in Ascomycota, is a powdery mildew parasite which is specific to *P. lanceolata*. Like all powdery mildews, *P. plantaginis* completes its life cycle as localized lesions on host tissue where it obtains nutrients via haustorial feeding roots. Infection causes significant stress for host plant, decreasing host growth and increasing host mortality (Laine, 2004; Susi & Laine, 2015). Almost 4,000 natural *P. lanceolata* populations located in the Åland islands, SW of Finland, have been surveyed annually since 2001 to study the metapopulation dynamics of *P. plantaginis* in this highly fragmented pathosystem (Jousimo et al., 2014). Between epidemic seasons, the parasite survives on decaying host leaves by producing overwintering structures, chasmothecia (Tack & Laine, 2014).

### 2.2 | Plant material

To produce genetically identical mother plants that differed in their infection status, we set up a common garden study in Lammi Biological station (Hämeenlinna, Finland), where cloned *P. lanceolata* plants were grown over years 2011–2013 with and without the pathogen *P. plantaginis* (Susi & Laine, 2015). Originally, seeds for the six mother plants were collected in 2010 from six distinct natural populations located in the Åland islands. The grown six mother genotypes were then propagated into altogether 51 individuals (2–14 clones representing each genotype; Figure S2a) and planted into common garden populations (Susi & Laine, 2015). Soil used in the experimental populations was a mixture of commercial soil substrate and sand (1:1), and the abiotic environmental conditions were same for all the plants. In this multi-year experiment, half of

the mothers were inoculated with two *P. plantaginis* strains (strain IDs L3 and L10, harvested from two allopatric populations in Åland islands) for three consecutive years. The infection level of each host plant was measured four times during every growing season through 2011–2013 (for more details, see Susi & Laine, 2015). We also measured the growth and reproduction of the host plants as number of leaves and flowers, respectively, first in July and secondly at the end of the growing season in September. The results of the analysis of this multi-year study are reported in Susi and Laine (2015), here we focus on reporting the findings regarding the six maternal genotypes that were included in this study.

After 3 years of repeated infection, we collected seeds from the six maternal genotypes, two clones of each (in total from 12 plants), where one was always heavily (more than 30% of leaves) infected (infected mothers) and another clone represented the same genotype, but was never infected (healthy mothers). From each of the 12 maternal plants, 18 seeds (half- or full-sibs, resulting from natural wind-pollination) were sown into 0.75 L pots. Half of the seeds ( $n = 9$ ) were sown into fertilized, nutrient-rich soil and the other half ( $n = 9$ ) to nutrient-poor soil (1:1 mixture of commercial soil substrate without added nutrients and sand). The plants in the nutrient-rich treatment received slow-release fertilizer twice, first portion (25 g of NPK 4-1-2 per plant) prior to the planting, and the second portion (20 g/plant) when we started the second common garden experiment (details below), in total 1.8 g of N, 0.45 g of P and 0.9 g of K. No fertilizer was added to the plants in the nutrient-poor treatment. Finally, the experiment consisted in total of 216 plants in four experimental groups: offspring in nutrient-rich or nutrient-poor soils originating from the healthy and infected mothers, 54 plants in each group. Seedlings were grown in the greenhouse conditions at  $20 \pm 2^\circ\text{C}$  (day) and  $16 \pm 2^\circ\text{C}$  (night) with 16:8 L:D photoperiod. When the seedlings were 18 days old, we moved them into National Plant Phenotyping Infrastructure (NaPPI, <https://www.helsinki.fi/en/infrastructures/national-plant-phenotyping>) platform.

### 2.3 | Early life measurements

We used the plant phenotyping facility located at the University of Helsinki, Viikki campus, for the phenotypic characterization of progeny's growth patterns. The plants were randomly arrayed into rows that were rotated every night to avoid microclimate differences influencing growing conditions. Plants were imaged every 72 hr by overhead CCD camera for Red Green Blue (RGB) images positioned in a PlantScreen™ analysis chamber (PSI, Czech R.). In addition, images from three sides (angles  $0^\circ$ ,  $120^\circ$  and  $240^\circ$ ) were taken simultaneously with overhead imaging. The images were obtained for all 216 plants in total 13 times during the 8-week period and pre-processed online as described in Awlia et al. (2016) to allow collecting RGB data. Obtained binary images from top view were used for calculating growth parameter of leaf area (PlantScreen™ analyzer, PSI, Czech R.), which has been found to yield precise data for several plant species (Humplík et al., 2015).

Because all three side angles (0°, 120° and 240°) resulted in similar estimations, the parameter 'height' only from angle 0° was included in the further analyses. The bolting day (a day when first flower stem was visible) and the number of developing flowers was manually observed and counted for each plant individual from the top RGB images.

## 2.4 | Common garden populations to measure offspring development over the growing season

To determine progeny growth and flowering traits under semi-natural conditions, we established a common garden population in the Viikki campus experimental field (Helsinki, Finland) after the phenotyping characterization. When the plants were 8-week old, they were acclimated for 1-week outdoors and then were planted into six common garden plots. In each plot, there were 36 plants of which half (18) of the plants originated from infected mothers, and half (18) from healthy mothers. Each maternal genotype was represented by three offspring from healthy plants, and three offspring from infected plants in each plot. The positions of the plants within plots were randomly assigned. To exclude the variation in the soil, the top layer (30 cm) was replaced with a mixture of commercial substrate without nutrients and sand (1:1) and the experimental area was covered with plastic to avoid weeds and asexual reproduction of the experimental plants. The plants in the nutrient-rich treatment (i.e. in the three experimental plots) received a second portion of fertilizer (20 g/plant of NPK 4-1-2) prior to planting. The experimental plots (three fertilized and three non-fertilized) were randomly arrayed in the experimental field. We counted the number of leaves, the length and width of the longest leaf, the number of flowers, the height of the longest stalk and the height of the inflorescence in the longest stalk, when the progeny plants were 16, 20 and 24 weeks old. After the last manual measurements, flower stalks were removed and the above-ground biomass was collected, dried and weighted.

## 2.5 | Inoculation experiment

To characterize the resistance of the progeny of infected and healthy mothers against powdery mildew *P. plantaginis*, we performed an inoculation experiment when the progeny plants were 24-weeks old. Leaves collected from 216 offspring plants were placed in random order on moist filter paper in Petri dishes and inoculated with conidial spores from purified powdery mildew lesions, by evenly brushing the spores on a leaf. We tested the resistance of the progeny plants against four *P. plantaginis* strains (L3, L10, 1630\_2 and 877), originally collected from natural populations in the Åland Islands. Lesion development was monitored under a dissecting microscope every second day, starting at day 3 and continued until day 13. For the statistical analysis, resistance was scored on day 13 as 0 = no resistance (infection) or 1 = resistance (no infection), when there was no

growth, or if the test plant showed rapid cell death (hypersensitive response) around the inoculum area.

## 2.6 | Statistical analysis

We ran all the analyses, including linear fixed effect models, in R software (R Development Core Team, 2016), using the `LME4` package (Bates et al., 2015). Each analysis was started with a full model with all the interactions included. Minimum adequate models were assessed through stepwise simplification and selection based on likelihood ratio or  $X^2$  values to compare significant interactions (Crawley, 2012). The effect of significant independent variables was derived from analysis of the minimum adequate model with the `CAR` package (Fox & Weisberg, 2011). Overdispersion was tested when necessary and accounted for by fitting an observation-level random effect. To detect the significant differences in resistance among genotypes in different nutrient treatments, a post hoc analysis was performed using the `MULTCOMP` package (Hothorn et al., 2008).

## 2.7 | Growth and reproduction of the mother plants

To test whether the maternal genotypes differed in their infection levels, we first ran a generalized linear model where we defined the proportion of diseased leaves ( $n = 25$  plants) at the highest peak of epidemics (July 2013) as the response variable, and genotype as categorical explanatory variables. To test whether the genotypes, and infected and healthy plants differed in their growth, we defined the number of leaves (in September 2013 count) as the response variable, and genotype and pathogen treatment (inoculation or no inoculation) as the categorical explanatory variables in the analysis. To understand the effect of infection and genotype on plant reproduction, we used the summed number of the flowers counted in July and September as the response variable, and genotype and pathogen treatment as the class explanatory variables. In models with two explanatory variables, the interaction of these variables was included in the model. In all models, we assumed a Poisson distribution of errors.

## 2.8 | Early growth and flower production of the progeny

To analyse the differences in early growth between nutrient treatment and maternal infection groups, we fitted mixed polynomial linear model (Mirman, 2014; Pavicic et al., 2017) for measurements obtained from the RGB images. The class explanatory variables treatment, maternal genotype, maternal infection and their interactions were fixed effects. The polynomial order of days after sowing was added as fixed effect to take into account the curved growth patterns over time. Plant ID was nested under a day and included as random effect to account for the repeated measurements. This structure was fitted to response variables area in mm<sup>2</sup> and height

computed from the top RGB images. To test whether experimental groups varied in their flower production, we analysed the number of flowers in every imaging day using a generalized linear mixed-effect model with log link and Poisson distribution of errors, with the same explanatory variables as above. The proportion of plants that were flowering (0/1) measured in every imaging day was analysed as a likelihood using a generalized linear model with a logit link function. The polynomial order of days and class explanatory variables treatment, maternal infection, maternal genotype and their interactions were defined as fixed effects.

## 2.9 | Growth and flower production of the progeny in the common garden study

To determine the differences between progeny groups among manually measured traits in the common garden experiment, we analysed the data from the last measuring point (24 weeks). The number of leaves and flowers was analysed as generalized linear mixed-effect models with a Poisson distribution of errors. Continuous response variables (the length and width of the longest leaf, the height of the tallest stalk, the height of inflorescence in the tallest stalk and the total biomass from dried plants) were analysed as linear mixed-effect models. The fixed class explanatory variables were nutrient treatment, maternal infection, maternal genotype and their interactions. The common garden plot was defined as a random effect in the models.

## 2.10 | Progeny resistance to powdery mildew measured through inoculations

To test whether maternal infection, genotype or offspring nutrient treatment affected the resistance responses against powdery mildew strains, we analysed the inoculation experiment data derived from laboratory observations. The inoculation response (0/1) on day 13 post-inoculation was modelled as a likelihood using a generalized

mixed-effect model with a logit link function. Maternal infection, genotype, offspring nutrient treatment and their interactions were fixed effects, and plant ID was nested under a mildew strain (the four mildew genotypes) as a random intercept.

## 3 | RESULTS

### 3.1 | Growth and reproduction of the mother plants

All the mother plants grown in common garden conditions with pathogen inoculation became heavily infected (33%–100% of the leaves infected). There were no significant differences among the genotypes in the proportion of infected leaves ( $p = 0.9126$ ; Tables S1 and S2; Figure S2a). We found that the mother plant genotypes varied in the number of leaves they produced ( $p < 0.0001$ ; Tables S1 and S2; Figure S2b), and that the effect of pathogen inoculation on growth was genotype specific ( $p < 0.0001$ ; Tables S1 and S2; Figure S2b). However, there was no direct significant effect of pathogen treatment on growth ( $p = 0.749$ ; Tables S1 and S2; Figure S2b). When we analysed the reproduction of the mother plants, we found that healthy plants generally produced more flowers than infected plants ( $96.9 \pm 5.6$  vs.  $77.1 \pm 5.2$ ;  $p < 0.0001$ ; Tables S1 and S2; Figure S2c). We also found a significant interaction between genotype and pathogen treatment ( $p < 0.0001$ ; Table S1; Figure S1c). Genotype alone had a significant effect on flower production ( $p < 0.0001$ ; Table S1; Figure S1c).

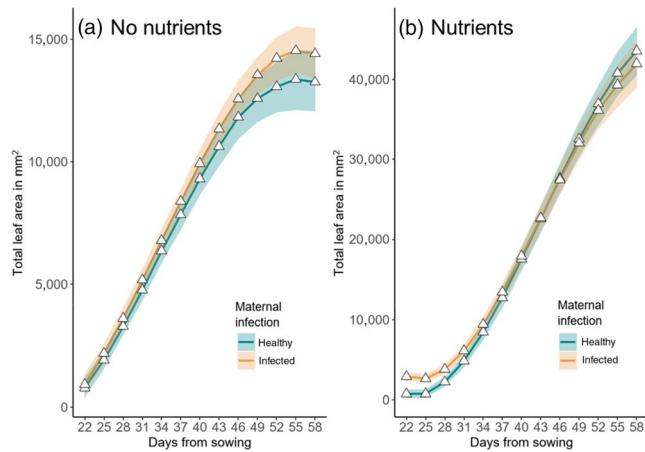
### 3.2 | Early growth and flower production of the progeny

To understand the differences in the progeny growth rates during the first 8 weeks, we compared the accumulation of the total leaf area ( $\text{mm}^2$ ) and height using the RGB imaging approach. The measured leaf area and plant height increased with time ( $p < 0.0001$ , Table 1; Figure 1; Table S3), and plants in the nutrient-rich conditions grew

**TABLE 1** Factors contributing to early growth of *Plantago lanceolata* plants in the greenhouse study analysed with polynomial linear mixed-effect models. Statistics for minimum adequate models with smallest AIC values are reported. Significant values are highlighted in bold

Source	Area ( $\text{mm}^2$ )		Height		Number of flowers		Flowering individuals	
	LRT	<i>p</i>	LRT	<i>p</i>	LRT	<i>p</i>	LRT	<i>p</i>
Days from sowing, polynomial order, <i>df</i> = 3	1,239.67	<b>&lt;0.0001</b>	961.68	<b>&lt;0.0001</b>	858.77	<b>&lt;0.0001</b>	21.64	<b>&lt;0.0001</b>
Nutrient treatment, <i>df</i> = 1	96.91	<b>&lt;0.0001</b>	19.28	<b>&lt;0.0001</b>	377.05	<b>&lt;0.0001</b>	3.16	0.07
Maternal infection, <i>df</i> = 1	16.38	<b>&lt;0.0001</b>	0.42	0.51	2.81	0.09	3.42	0.06
Genotype, <i>df</i> = 5	9.24	0.099	9.63	0.086	6.07	0.2	15.62	<b>0.008</b>
Nutrient treatment $\times$ maternal infection, <i>df</i> = 1	10.21	<b>0.001</b>	6.09	<b>0.01</b>	12.99	<b>0.0003</b>	1.54	0.21
Random	ID	Days	ID	Days	ID	Days	ID	Days
Variance	159,622,863	251,066	23,784.8	32.85	22.76	0.03	0.01	18.38
St. dev.	12,634.20	501.10	154.22	5.73	4.77	0.16	0.11	4.29

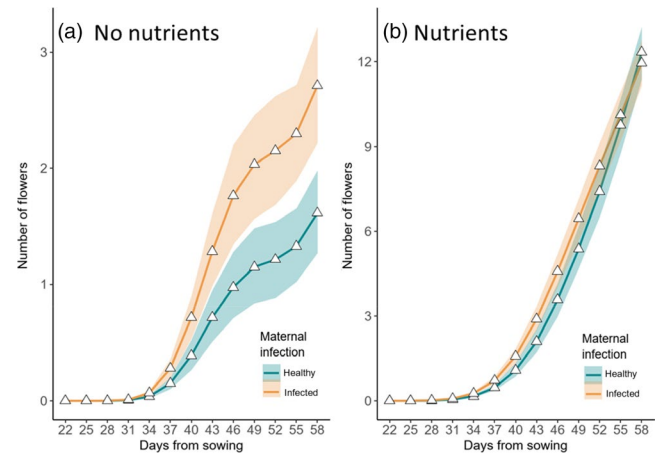




**FIGURE 1** Development of detected top leaf area ( $\text{mm}^2$ ) from RGB images. Plant early development between first 22–58 days as measured in pixels and transferred into  $\text{mm}^2$ , detected by RGB imaging. Growth of progeny of healthy and infected mothers was followed in two treatments, where (a) no nutrients were added and (b) nutrients were added. Curves and confidence intervals from fitted models,  $n = 54$  plants per treatment and maternal infection group. The triangles represent the average for each group at a given time point

faster and produced more leaves than the plants in the nutrient-poor conditions ( $p < 0.0001$ , Table 1; Figure 1; Table S3). The progeny of infected mothers produced more leaf area ( $p < 0.0001$ , Table 1; Figure 1; Table S3), especially in the nutrient-poor treatment (interaction between nutrition treatment and maternal infection,  $p < 0.0001$ , Table 1; Figure 1a; Table S3). Maternal genotype had no effect on the detected leaf area ( $p = 0.09$ , Table 1) or height ( $p = 0.51$ , Table 1). Plant height was affected by both the nutrient treatment ( $p < 0.0001$ , Table 1), and a significant interaction between nutrient treatment and maternal infection ( $p = 0.01$ , Table 1; Table S3).

To understand how maternal infection and nutrient treatment affect offspring flower production over the first 8 weeks, we quantified the number of flowers from top RGB images. As expected, the proportion of plants that were flowering, and the number of flowers increased with time ( $p < 0.0001$  and  $p < 0.0001$ , respectively, Table 1; Figure 2; Table S2). In the last imaging round on day 58, 67% of progeny of healthy mothers and 81% of progeny of infected mothers were flowering in the nutrient-poor treatment, and 96% of healthy and 98% of progeny of infected mothers were flowering in the nutrient-rich treatment (nutrient treatment,  $p = 0.07$ , Table 1; Table S3). Maternal infection had no significant effect on the proportion of flowering plants ( $p = 0.06$ , Table 1), but the genotypes differed in their flowering efforts ( $p = 0.008$ ; Table 1). In general, plants produced significantly more flowers in the nutrient-rich treatment than in the nutrient-poor treatment ( $p < 0.0001$ , Table 1; Figure 2; Table S3). Neither maternal infection nor genotype had a significant effect on the number of flowers produced (infection  $p = 0.09$ , genotype  $p = 0.2$ , Table 1; Table S3). However, we found a significant interaction between nutrient treatment and maternal infection for the number of flowers produced. The progeny of infected mother



**FIGURE 2** Flower development in the progeny of healthy and infected mothers. Number of flowers detected from top RGB images through early life in two treatments, where (a) no nutrients were added and (b) nutrients were added. Curves from fitted models with 95% confidence intervals,  $n = 54$  plants per treatment and maternal infection group. The triangles represent the average for each group at a given time point

produced more flowers in the nutrient-poor treatment, while in the high nutrient treatment flower production was similar between the progeny of both infected and healthy mothers (interaction between nutrient treatment and maternal infection,  $p = 0.0003$ , Table 1; Figure 2; Table S2).

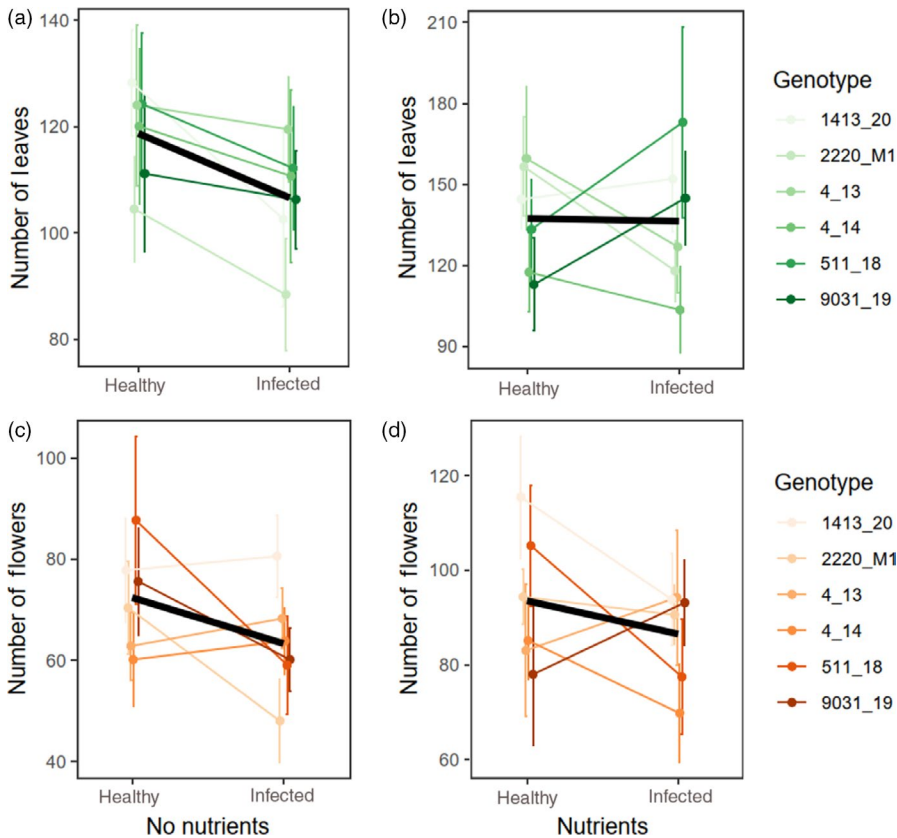
### 3.3 | Growth and flower production of the progeny in the common garden study

To understand how maternal infection and nutrient treatment affect vegetative growth and the number of flowers produced in 8-week-old to 24-week-old progeny, we measured phenotypic traits manually in common garden populations. The phenotypic measurements conducted at the last time point revealed that the accelerated growth and reproduction rate detected in the progeny of infected mothers during early life were not detected anymore when the plants were 24 weeks old (Figure S3). By the last manual measurement time point in early September, offspring of infected mothers had produced less flowers and leaves than the offspring of healthy mothers (maternal infection  $p < 0.0001$  and  $p < 0.0001$ , respectively; Table 2; Figure 3; Table S4). Plants produced more flowers and leaves in the nutrient-rich treatment (Table 2; Figure 3), and the genotypes also differed in their flower and leaf production ( $p < 0.0001$  and  $p < 0.0001$ , respectively; Table 2; Figure 3; Table S4). We found a significant three-way interaction between maternal infection, nutrient treatment and maternal genotype on both the number of flowers and leaves produced ( $p < 0.0001$  and  $p < 0.0001$ , respectively, Table 2; Figure 3; Table S4). These significant three-way interactions indicate that the effects of maternal infection on leaf size and flower production vary depending on the maternal genotype and the nutrient conditions that the progeny encounter.

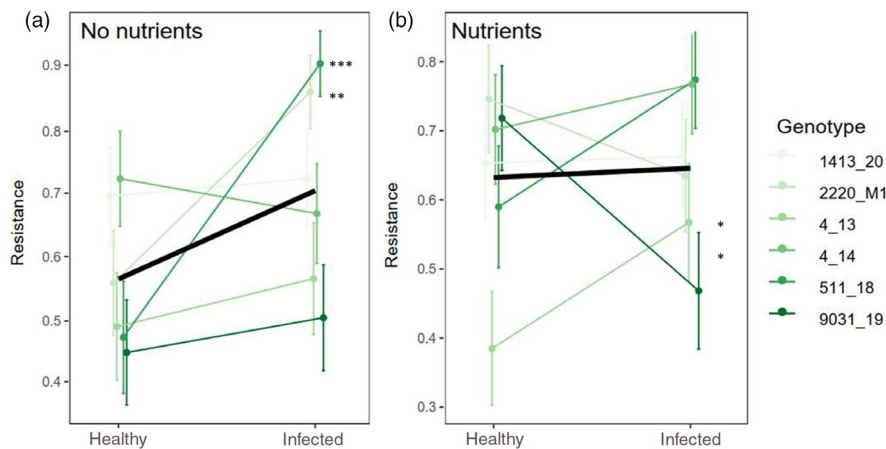
**TABLE 2** Factors contributing to the growth and resistance of progeny plants in the common garden study and in the laboratory experiment, analysed with linear mixed-effect models. Statistics for minimum adequate models with smallest AIC values are reported. Significant values are highlighted in bold

Source	No. of leaves		No. of flowers		Longest leaf		Leaf width		Longest stalk		Inflorescence		Biomass		Resistance	
	LRT	p	LRT	p	LRT	p	LRT	p	LRT	p	LRT	p	LRT	p	LRT	p
Nutrient treatment, <i>df</i> = 1	12.93	<b>0.0003</b>	6.22	<b>0.01</b>	—	<i>ns</i>	—	<i>ns</i>	12.90	<b>0.0003</b>	7.87	<b>0.005</b>	5.39	<b>0.02</b>	0.12	0.72
Maternal infection, <i>df</i> = 2	19.12	<b>&lt;0.0001</b>	42.24	<b>&lt;0.0001</b>	—	<i>ns</i>	2.78	0.095	0.13	0.714	—	<i>ns</i>	—	<i>ns</i>	3.77	<b>0.049</b>
Genotype, <i>df</i> = 5	129.35	<b>&lt;0.0001</b>	127.77	<b>&lt;0.0001</b>	11.35	<b>0.044</b>	5.25	0.386	5.88	0.318	11.48	0.04	—	<i>ns</i>	25.03	<b>&lt;0.001</b>
Maternal infection × Nutrient treatment, <i>df</i> = 1	16.99	<b>&lt;0.0001</b>	2.86	0.09	—	<i>ns</i>	—	<i>ns</i>	—	<i>ns</i>	—	<i>ns</i>	—	<i>ns</i>	—	<i>ns</i>
Maternal infection × Genotype, <i>df</i> = 5	96.19	<b>&lt;0.0001</b>	85.68	<b>&lt;0.0001</b>	—	<i>ns</i>	14.45	<b>0.013</b>	24.75	<b>0.0001</b>	11.9	0.03	—	<i>ns</i>	—	<i>ns</i>
Genotype × Nutrient treatment, <i>df</i> = 5	97.24	<b>&lt;0.0001</b>	23.90	<b>0.0002</b>	—	<i>ns</i>	—	<i>ns</i>	—	<i>ns</i>	—	<i>ns</i>	—	<i>ns</i>	—	<i>ns</i>
Maternal infection × Genotype × Nutrient treatment, <i>df</i> = 5	77.96	<b>&lt;0.0001</b>	68.36	<b>&lt;0.0001</b>	—	<i>ns</i>	—	<i>ns</i>	—	<i>ns</i>	—	<i>ns</i>	—	<i>ns</i>	11.31	<b>0.04</b>
Random (plot)																
Variance	0.00	0.02	6.18	—	0.05	—	3.24	—	0.02	—	18.8	—	1.34	—	0.30	—
St. dev.	0.07	0.14	2.49	—	0.23	—	1.8	—	0.16	—	4.34	—	1.15	—	0.54	—





**FIGURE 3** Total number of leaves and flowers produced at the end of growing season. Reaction norms for number of leaves in progeny of healthy and infected mothers by genotype in two treatments, where (a) no nutrients were added and (b) nutrients were added. Number of flowers in (c) no nutrients (d) nutrients treatment by genotype. The mean values for reaction norms in each nutrient treatment–maternal infection groups are shown by black lines. Standard errors for each genotype are shown



**FIGURE 4** Mean resistance of progeny of six *Plantago lanceolata* maternal genotypes in nutrient-poor and nutrient-rich treatments. Reaction norms for resistance in the progeny of healthy and infected mothers in two treatments, where (a) no nutrients were added and (b) nutrients were added. Resistance was tested against four *Podosphaera plantaginis* strains. The mean values for reaction norms in both nutrient treatment–maternal infection groups are shown by black lines. Standard errors for each genotype are shown. Significant interactions among genotypes are indicated by asterisks

For the other measured life-history traits, nutrient treatment was the most significant factor affecting growth ('Nutrient treatment' row in Table 2; Figure S3). Total growth was highest in the high nutrient treatment for the following measurements: number of leaves (on average 111.2 in nutrient-poor and 136.4 in nutrient-rich treatment), number of flowers (68.4 and 89.3), longest stalk (42.6 and 49.1 cm), largest inflorescence size (4.3 and 4.9 cm) and dried biomass (19.6 and 28.6 g), respectively (Figure S3). The length of the longest leaf (on average 24.4 cm) was defined by the maternal genotype, and the leaf width (2.3 cm) and the longest stalk (47.3) by the interaction between maternal genotype and infection (Table 2; Figure S3).

### 3.4 | Progeny resistance to powdery mildew measured through inoculations

To study how maternal infection and nutrient treatment affect progeny disease resistance, we characterized the progeny phenotypic resistance against powdery mildew *P. plantaginis* strains in a laboratory inoculation experiment when the progeny were 24 weeks old. On day 13 post-inoculation, 36.3% of the leaves showed infection. The resistance phenotype of the progeny was significantly affected by maternal genotype ( $p < 0.001$ , Table 2; Figure 4; Table S4). There was no significant difference between the detected resistance responses of progeny grown in nutrient-rich or nutrient-poor

conditions (Table 2,  $p = 0.72$ , Figure 4; Table S4). In general, there was a tendency for progeny of infected mothers to be more resistant than the progeny of healthy mothers ( $p = 0.049$ , Table 2; Figure 4; Table S4). However, measured levels of resistance varied significantly according to genotype and nutrient as shown by a significant three-way interaction between maternal nutrient treatment, maternal infection and genotype ( $p = 0.04$ , Table 2; Figure 4). This significant three-way interaction indicates that the effects of maternal infection on progeny resistance vary depending on the mother plant genotype and nutrient conditions that the progeny encounter.

## 4 | DISCUSSION

The extensive variation in disease resistance that natural populations support (Laine et al., 2011) has been traditionally considered to reflect genetic diversity. However, recent studies have highlighted that the effects of pathogen infection may transcend generations also via non-genetic mechanisms (Holeski et al., 2012; Martinez-Medina et al., 2016; Mauch-Mani et al., 2017; Singh & Roberts, 2015). Pathogen infection in a perennial, long-living plant may affect offspring fitness and resistance phenotype, yet its extent remains largely unknown. Here, we determined how maternal infection affects progeny fitness and resistance in contrasting nutrient environments. Our results reveal that the negative effect powdery mildew infection has on the mother plants also transcends to impact their progeny. By the end of the growing season, the progeny of infected mothers had grown less, and produced fewer flowers than the progeny of healthy mothers. The offspring of infected mothers also had a tendency to have higher resistance against infection by *P. plantaginis*. Moreover, we discovered that maternal infection affected progeny development and resistance both directly, as well as through a three way-interaction between maternal genotype, maternal infection status and nutrient availability.

In line with previous studies (Laine, 2004; Penczykowski et al., 2015), we found a negative effect of infection on the maternal plant generation in the common garden study where plants were grown with and without infection. The negative effect of *P. plantaginis* on the growth of *P. lanceolata* was genotype specific while flower production was negatively affected by infection both directly and via genotype-specific effects. Indeed, there is a strong evidence showing that pathogen infection may reduce host fitness and the quantity and quality of produced seeds (Agrios, 2005; Argyris et al., 2003). Asexual reproduction may also be altered by pathogen infection, as shown in *Trientalis europea* where plants infected by the systemic smut *Urocystis trientalis* produced smaller ramets than healthy plants (Piqueras, 2001). Hence, it is not surprising that the effects of infection may transcend to affect a wide range of life-history traits also in the progeny. We found that both infected mothers and their non-infected offspring produced fewer flowers than the healthy mothers and their offspring. Similarly, progeny of wheat plants infected by powdery mildew *Blumeria graminis* produced fewer and lighter seeds in two generations after an epidemic (Jarosz et al., 1989), and

ryegrass *Lolium multiflorum* progeny produced smaller and less vigorous seeds when their parents were infected by a rust pathogen *Puccinia coronata* (Mattner & Parbery, 2007).

We found that the offspring of mothers that had experienced powdery mildew infection had adapted a life-history strategy that favours early reproduction. Initially, in the greenhouse study, the size of progeny plants—measured as leaf area—grew faster if the mother had been infected. Similarly, in their study, Latzel et al. (2009) showed that *Plantago* plants produced more and longer leaves, and had higher photosynthetic capacity if mothers were mechanically disturbed and grown in stressful nutrient-poor environments, even in their second year (Latzel et al., 2010). In our study, the progeny of infected mothers not only grew faster but also bolted earlier in both nutrient treatments. Increased abiotic or biotic stress is known to accelerate flowering in many plant species (Banday & Nandi, 2015; Brachi et al., 2012; Cho et al., 2017). As the differences in progeny growth and flowering patterns due the maternal infection were evidenced mostly in nutrient-poor conditions similar to those experienced by the mother plants, our results are in line with previous studies suggesting that maternal effects are responsible for enhanced growth of progeny in maternal conditions (Donohue & Scmitt, 1998; Galloway, 2005), and the maternal stress results in enhanced growth in progeny (Latzel et al., 2009; Sultan, 1996; Yin et al., 2019). However, we found the opposite effect of maternal infection later in the growing season with progeny of infected mothers being smaller and producing fewer flowers than those of healthy mothers. The epidemic of *P. plantaginis* in Åland islands does not peak until August (Ovaskainen & Laine, 2006) while flowering starts in early June. Hence, this strategy would allow individuals to maximize their fitness when risk of infection is expected to be high.

We found that the progeny of infected *Plantago* mother clones had a tendency to be more resistant to powdery mildew infection. Both the strength and the direction of this trend depended on maternal genotype and nutrient availability. Increasing evidence has shown that pathogens can prime defence plasticity in plants, and that the enhanced resistance may be transgenerational (Holeski et al., 2012; Mauch-Mani et al., 2017). The mechanisms underpinning pathogen-induced epigenetic changes have been detected in several annual species such as *Arabidopsis* (reviewed by Martinez-Medina et al., 2016; Mauch-Mani et al., 2017). Since the discovery that defence priming can be transmitted to future generations, similar effects have been described in cultivated crops (Walters et al., 2013) and in legumes (Martinez-Aguilar et al., 2016; terHorst & Lau, 2012). We tested priming effects after three consecutive years of maternal infection, which may have strengthened the detected resistance responses in the progeny. Because our study spans only one growing season of progeny growth and reproduction, we cannot determine the durability of increased resistance over multiple seasons or generations. However, a study conducted in natural populations of the *Plantago-Podosphaera* system has revealed that resistance is higher in areas where disease encounter rates have been high compared to areas where it has been low (Laine, 2006). This phenomenon could have arisen both via direct genetic changes in these populations or

via transgenerational priming. An exciting future avenue of research would be to determine the extent to which transgenerational priming contributes to the high levels of phenotypic resistance diversity observed in natural populations (Laine et al., 2011).

Importantly, we found evidence for a strong direct genotype effect on disease resistance, and both direct and genotype-by-maternal infection interactions on progeny growth, and reproduction. The strong genotypic differences in disease resistance detected here are in line with earlier studies on *P. lanceolata* (Laine, 2004, 2007). Overall, genotype-specific responses in the progeny to maternal infection may be an important mechanism generating phenotypic variation in these key life-history traits within and among the local populations of *P. lanceolata*. Similarly, maternal effects under herbivory stress in long-living *Populus* sp. varied over genotypes, and offspring differed significantly in their constitutive allocation to growth and resistance traits (Holeski et al., 2013). In the clonal species, for example, in *Trifolium repens*, the direction and strength of maternal effects on offspring biomass were altered positively or negatively depending on the type of abiotic maternal stress, and these effects were highly genotype specific (Rendina González et al., 2018).

A recent meta-analysis (Yin et al., 2019) found that while transgenerational effects often enhance offspring performance in response to both stressful and benign conditions, perennial plants show hardly any transgenerational responses at all, suggesting other strategies for adaptation. However, it should be noted that there are relatively few case studies on perennial plants (21 studies were identified in meta-analysis by Yin et al., 2019), and these have generally not accounted for the high genotypic and environmental variation typical of natural plant populations. The three-way interactions we find between maternal infection, genotype and nutrient availability highlight that maternal effects on progeny fitness and disease resistance are not consistent across genotypes and environments. Our results suggest that the advantage of transgenerational defence priming occurs when environmental conditions become more challenging (Kuijper & Hoyle, 2015; Reiss & Drinkwater, 2018). Overall, fine-scale variation in genetic diversity and environmental conditions typical of natural plant populations are likely to amplify the extent to which maternal effects may generate variation in offspring quality both within and between populations. Our results are in line with findings by Gáspár et al. (2019), who discovered that epigenetic differences across *P. lanceolata* populations were consistently related to genetic and environmental variation. Although the mechanistic underpinning maternal effects of infection in *P. lanceolata* is not known, the strong phenotypic responses we find are compelling evidence of the role that maternal effects in heterogeneous environments can have in generating phenotypic diversity in perennial plants.

Jointly, our results support the idea that phenotypic variation within a generation stems not only from the genetic inheritance of parental alleles but can also be adjusted by maternal effects in response to abiotic and biotic stress experienced by the parental generation (Auge et al., 2017). Overall, our results indicate that maternal infection may influence progeny growth, flower production and phenotypic

resistance diversity in long-lived plant species. Both empirical and theoretical work have shown that variation in resistance to natural enemies across spatially structured populations can fundamentally alter epidemiological and evolutionary patterns of infectious disease (Laine et al., 2011; Salvaudon et al., 2008; Tack et al., 2012), yet to our knowledge this is the first study to demonstrate how the effects of maternal infection on progeny resistance vary depending on the mother plant genotype and nutrient conditions that the progeny encounter. This work provides novel insights into how present conditions may shape future generations of perennial plants to help them cope with the same stressors as their parental generation in an adaptive manner.

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## AUTHORS' CONTRIBUTIONS

A.-L.L., L.H. and H.S. conceived the ideas and designed the experiment; L.H. and H.S. conducted the experimental work and L.H. analysed the data; L.H. and A.-L.L. led the writing of the manuscript; all the authors contributed to the drafts and gave final approval for publication.

## DATA AVAILABILITY STATEMENT

Data available from the Dryad Digital Repository <https://doi.org/10.5061/dryad.0zpc866wm> (Höckerstedt et al., 2020).

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## SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section.

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